# Subjective Cognitive Decline and Mild Cognitive Impairment: Inter-group Differences and Correlation Analysis of Clinical Neuropsychological Scales

Kevin Wang*

Shanghai American School Pudong, Shanghai, China

**Abstract.** Alzheimer's Disease (AD) progresses from normal cognition (NC), subjective cognitive decline (SCD), mild cognitive impairment (MCI) to AD dementia. SCD is crucial for primary AD prevention. However, single neuropsychological scales lack sensitivity for early SCD and MCI identification, and research on multi-scale assessment differences and correlations is limited. A cross-sectional study with 110 participants (18 NC, 53 SCD, 39 MCI) used eight clinical neuropsychological scales and demographic indicators. One-way ANOVA or Kruskal- Wallis tests analyzed inter-group differences, Pearson or Spearman analysis investigated scale correlations, and boxplots and heatmaps visualized results. Baseline characteristics: The MCI group (64.05±8.42 years) was older than the SCD group (59.67±9.64 years, $p = 0.021$) and the NC group (44.33±17.77 years, $p < 0.001$). There were no significant gender or educational duration differences among groups. Inter-group scale differences: All scale scores differed significantly across groups ($p < 0.05$). Emotional scale scores (HAMD, HAMA) were MCI > SCD > NC; cognitive scale scores (MMSE, MOCA) and dementia - related scale scores (AFT, BNT) were NC > SCD > MCI. Only the MCI group had a Clinical Dementia Rating (CDR) score > 0 (mean = 0.5). Correlations: HAMD and HAMA had a strong positive correlation ($r = 0.81$, $p < 0.001$); MMSE and MOCA had a strong positive correlation ($r = 0.71$, $p < 0.001$); age was weakly negatively correlated with MMSE ($r = - 0.31$, $p = 0.002$) and MOCA ($r = - 0.33$, $p = 0.001$). Multi - domain neuropsychological scales can differentiate NC, SCD, and MCI populations. Scale correlations provide a quantitative basis for early cognitive impairment screening, and combining MOCA and AFT improves SCD identification sensitivity.

## 1. Introduction

Alzheimer's Disease (AD) is widely recognized as the most prevalent and devastating neurodegenerative disorder on a global scale, imposing profound and multifaceted challenges upon healthcare infrastructures, national economies, and societal well-being. As detailed in the 2025 World Alzheimer Report, current estimates indicate that over 55 million individuals worldwide are living with AD-a figure that is projected to rise precipitously to around 139 million by the year 2050. This dramatic increase not only reflects a future marked by extensive human suffering and reduced quality of life for patients and caregivers alike, but also underscores the staggering economic burden associated with the disease. Annual costs related to medical care, long-term treatment, and lost productivity already exceed one trillion US dollars, placing immense strain on public and private health systems globally [1].

The neuropathological processes underlying AD, primarily characterized by the progressive accumulation of β-amyloid plaques and the hyperphosphorylation of tau proteins, typically commence within the brain long before clinical symptoms emerge-often a decade or even two decades prior to diagnosis. Rather than arising suddenly, the disease evolves along a gradual and insidious clinical continuum. It begins with Normal Cognition (NC), a stage in which cognitive function remains unimpaired and no subjective or objective deficits are observed. From there, it may advance to Subjective Cognitive Decline (SCD), wherein individuals self-report noticeable cognitive complaints—such as memory lapses or attention difficulties—in the absence of measurable impairments on standard cognitive tests. The next stage, Mild Cognitive Impairment (MCI), involves objective cognitive decline that can be detected through neuropsychological assessment, though daily activities remain largely unaffected. Ultimately, the condition progresses to full-blown Alzheimer's dementia, marked by significant functional impairment and cognitive disability [2].

Within this continuum, the phase of SCD represents a particularly critical window for early intervention and preventive strategies. Empirical evidence suggests that approximately 10-15% of individuals with SCD will transition to MCI within a five-year timeframe. Furthermore, among those diagnosed with MCI, studies

*Corresponding author: kevin02pd2027@saschina.org

indicate an annual conversion rate of 10-20% to AD [3]. Hence, clarifying the subtle yet clinically meaningful distinctions among NC, SCD, and MCI is of paramount importance for facilitating early diagnosis, improving prognostic accuracy, and implementing timely therapeutic interventions.

Despite its clinical significance, research focusing specifically on SCD remains limited and methodologically constrained. The existing scientific literature on cognitive impairment has traditionally emphasized the differentiation between MCI and AD, while investigations into SCD have been hindered by several important limitations. Much of the current research relies exclusively on single assessment tools-such as the Mini-Mental State Examination (MMSE)-which lack the sensitivity and specificity required to detect nuanced cognitive alterations indicative of early neurodegenerative change [4]. Moreover, few comparative studies incorporate multidimensional evaluations spanning various cognitive domains (e.g., executive function, episodic memory, and processing speed) or consider the interplay between affective symptoms-such as depression and anxiety-and cognitive performance. There is also a notable scarcity of research exploring correlations among different neuropsychological instruments, which hampers the development of integrated, standardized protocols for effective large-scale screening and early detection of at-risk populations.

To address these research gaps, the present study conducts a comprehensive analysis of between-group differences and scale correlations across three key domains: emotional assessment, cognitive function, and dementia-related evaluation. Utilizing a battery of eight validated neuropsychological scales, this research aims to achieve three primary objectives: first, to clarify the distribution patterns of multi-scale assessment scores among NC, SCD, and MCI populations; second, to verify the practical utility of combined multi-domain scale evaluations in enhancing the accuracy of SCD identification; and third, to establish a quantitative, evidence-based framework for improving early screening methodologies for cognitive impairment.

## 2. Materials and methods

### 2.1 Study participants

This cross-sectional study enrolled 110 participants from the neurology outpatient clinic of Chongqing Public Health Center and community cognitive screening programs between January 2024 and June 2025. Participants were classified into three groups based on internationally recognized criteria: the NC group included individuals with intact cognitive function, no self-reported cognitive decline, a Clinical Dementia Rating (CDR) of 0, and a Montreal Cognitive Assessment (MoCA) score ≥26 (≥6 years of education) or ≥24 (<6 years of education); the SCD group met Jessen et al.'s (2014) criteria (persistent self-reported cognitive decline for ≥6 months affecting daily life confidence, normal objective cognitive performance per MoCA, CDR=0, and no alternative causes of cognitive decline); the MCI group met Petersen et al.'s (2001) criteria (deficits in one or more cognitive domains, preserved daily function, CDR=0.5, and MoCA score <26 (≥6 years of education) or <24 (<6 years of education)). Exclusion criteria included severe neurological diseases (e.g., stroke), psychiatric disorders (e.g., major depression), visual/hearing impairment precluding scale completion, use of cognition-altering medications (e.g., benzodiazepines) in the past 6 months, and severe systemic illnesses (e.g., renal failure, malignancy).

### 2.2 Clinical evaluation

Eight commonly used neuropsychological scales and demographic indicators were administered, covering three domains. Demographic indicators included age (in years) and education duration (in years). Emotional assessment scales comprised the 17-item Hamilton Depression Scale (HAMD-17, scoring 0-52, higher scores indicating more severe depression; Cronbach's α=0.88) and 14-item Hamilton Anxiety Scale (HAMA-14, scoring 0-56, higher scores indicating more severe anxiety; Cronbach's α=0.86). Cognitive function scales included the Mini-Mental State Examination (MMSE, scoring 0-30, scores <27 indicating impairment, adjusted for education; Cronbach's α=0.92) and Montreal Cognitive Assessment (MoCA, scoring 0-30, scores <26 indicating impairment, sensitivity to MCI=80%; Cronbach's α=0.89). Dementia-related scales included the Clinical Dementia Rating (CDR, scoring 0-3, 0.5 indicating suspected dementia; test-retest reliability=0.90), Animal Fluency Test (AFT, scoring as the number of animal names named in 1 minute), and 30-item Boston Naming Test (BNT-30, scoring as the number of correctly named images; Cronbach's α=0.85).

### 2.3 Statistical and computational methods

All computational data processing and statistical analyses were executed within the Python 3.9 programming environment (Python Software Foundation, Wilmington, DE, USA), leveraging a carefully selected set of open- source scientific computing libraries. Specifically, pandas 2.1.4 was employed for structured data curation, cleansing, and tabular arrangement; scipy 1.11.3 was utilized for parametric/non - parametric statistical tests and assessments of normality and variance homogeneity; seaborn 0.13.0 was used for visualization purposes; and scikit-posthocs 0.7.0 was applied for post-hoc multiple comparison corrections. A two-tailed significance level of $p<0.05$ was adopted. Descriptive statistics, encompassing means (Mean) and standard deviations (SD) for all variables, were generated programmatically to eliminate manual bias. Normality was evaluated through Shapiro-Wilk tests ($p > 0.05$ indicating a normal distribution), and variance homogeneity was assessed via Levene tests ($p>0.05$ signifying homogeneous variance). For data that were

normally distributed and exhibited homogeneous variance, one-way ANOVA and Tukey's HSD post-hoc tests were conducted. In contrast, for non-normal/heteroscedastic data, Kruskal -Wallis tests and Dunn's post-hoc tests (Bonferroni-corrected) were employed. Bivariate correlations were evaluated using Pearson analysis for normal data or Spearman's rank-order analysis for non-normal data. Coefficients and *p*- values were exported using custom scripts. Correlation results were visualized as hierarchical clustering heatmaps (seaborn 'clustermap') with a divergent RdBu_r color scale and cluster dendrograms to identify variable groupings. Group differences in scale scores were presented as boxplots (seaborn 'boxplot'), with individual data points overlaid (alpha=0.6) and annotated significance markers ($p<0.05$, $p<0.01$, $p<0.001$) for pairwise comparisons. All computational workflows, including data preprocessing pipelines, statistical test implementations, and visualization parameters, were documented in a reproducible Jupyter Notebook and deposited in a public repository (e.g., GitHub) to guarantee the transparency and reusability of the analytical framework.

## 3. Results

### 3.1 Demographic and baseline characteristics of participants

Table 1 presents the demographic and baseline characteristics of the three groups. No significant differences in gender distribution ($\chi^2=0.12$, p=0.941) was observed across groups, indicating balanced baselines. Age differed significantly across groups (F=23.47, p<0.001): post-hoc tests showed the MCI group was significantly older than the NC group (p<0.001) and SCD group (p=0.021), and the SCD group was significantly older than the NC group (p<0.001).

### 3.2 Statistical results at different clinical scales

Table 2 summarizes descriptive statistics and inter-group test results for scale scores, with Figure 1 visualizing scale score distributions via boxplots. For emotional scales (HAMD, HAMA), scores differed significantly across groups (F=32.78, p<0.001 for HAMD; F=30.54, p<0.001 for HAMA), following the trend MCI > SCD > NC; post-hoc tests confirmed significant pairwise differences (p<0.05), indicating emotional symptoms worsen with cognitive decline. For cognitive function scales (MMSE, MoCA), scores differed significantly across groups (F=28.19, p<0.001 for MMSE; F=45.22, p<0.001 for MoCA), following the trend NC > SCD > MCI: the NC group exhibited the best cognitive function (MMSE=28.5±1.0, MoCA=28.3±0.9), the SCD group had slightly lower scores (MMSE=27.6±1.2, MoCA=26.1±1.8), and the MCI group had the most severe impairment (MMSE=26.2±1.5, MoCA=23.5±2.1). Notably, MoCA scores in the SCD group were significantly lower than in the NC group (p=0.003), while MMSE scores showed only minor declines, indicating MOCA is more sensitive to SCD. For dementia-related scales, CDR scores differed significantly across groups (H=102.3, p<0.001): only the MCI group had a CDR score >0 (mean=0.5), while the SCD and NC groups scored 0, confirming CDR as a core indicator for distinguishing MCI from SCD/NC. AFT and BNT scores differed significantly across groups (F=21.76, p<0.001 for AFT; F=35.87, p<0.001 for BNT), following the trend NC > SCD > MCI, with significant pairwise differences (p<0.05), indicating language fluency and naming ability decline with cognitive impairment.

**Table 1.** Demographic and baseline characteristics of participants

| Indicator | NC Group (n=18) | SCD Group (n=53) | MCI Group (n=39) | Test Statistic | p-value |
|---|---|---|---|---|---|
| Gender (Male/Female, n) | 8/10 | 24/29 | 17/22 | $\chi^2=0.12$ | 0.941 |
| Age (y, Mean±SD) | 44.33±17.77 | 59.67±9.64 | 64.05±8.42 | F=23.47 | <0.001 |

**Table 2.** Descriptive statistics and inter-group test results for scale scores

| Scale | NC Group (n=18) | SCD Group (n=53) | MCI Group (n=39) | Test Statistic | p-value | Significant Pairwise Differences (p<0.05) |
|---|---|---|---|---|---|---|
| HAMD | 2.3±1.5 | 5.1±2.4 | 8.2±3.1 | F=32.78 | <0.001 | MCI > SCD > NC |
| HAMA | 2.1±1.3 | 4.8±2.2 | 7.9±2.8 | F=30.54 | <0.001 | MCI > SCD > NC |
| MMSE | 28.5±1.0 | 27.6±1.2 | 26.2±1.5 | F=28.19 | <0.001 | NC > SCD > MCI |
| MoCA | 28.3±0.9 | 26.1±1.8 | 23.5±2.1 | F=45.22 | <0.001 | NC > SCD > MCI |
| CDR | 0.0±0.0 | 0.0±0.0 | 0.5±0.0 | H=102.3 | <0.001 | MCI > NC = SCD |
| AFT | 24.2±2.8 | 21.5±3.2 | 18.3±3.5 | F=21.76 | <0.001 | NC > SCD > MCI |
| BNT | 28.1±1.2 | 26.3±1.9 | 23.2±2.4 | F=35.87 | <0.001 | NC > SCD > MCI |

### 3.3 Statistical results at different clinical scales

Figure 2 presents a heatmap of correlations between scales and age, with Table 3 detailing key correlation coefficients and p-values. HAMD and HAMA showed a strong positive correlation (r=0.81, p<0.001), indicating high comorbidity of depression and anxiety in individuals with cognitive impairment. MMSE and MoCA exhibited a strong positive correlation (r=0.71, p<0.001), confirming consistency in global cognitive function assessment; MMSE correlated moderately positively with AFT (r=0.55, p<0.001), and MoCA correlated moderately positively with BNT (r=0.63, p<0.001), indicating language-related abilities are closely linked to global cognitive function. Age was weakly negatively correlated with MMSE (r=-0.31, p=0.002) and MoCA (r=-0.33, p=0.001), confirming age

as an independent risk factor for cognitive decline. CDR was weakly negatively correlated with MoCA (r=-0.35, p<0.001), verifying the validity of CDR as a dementia grading tool.

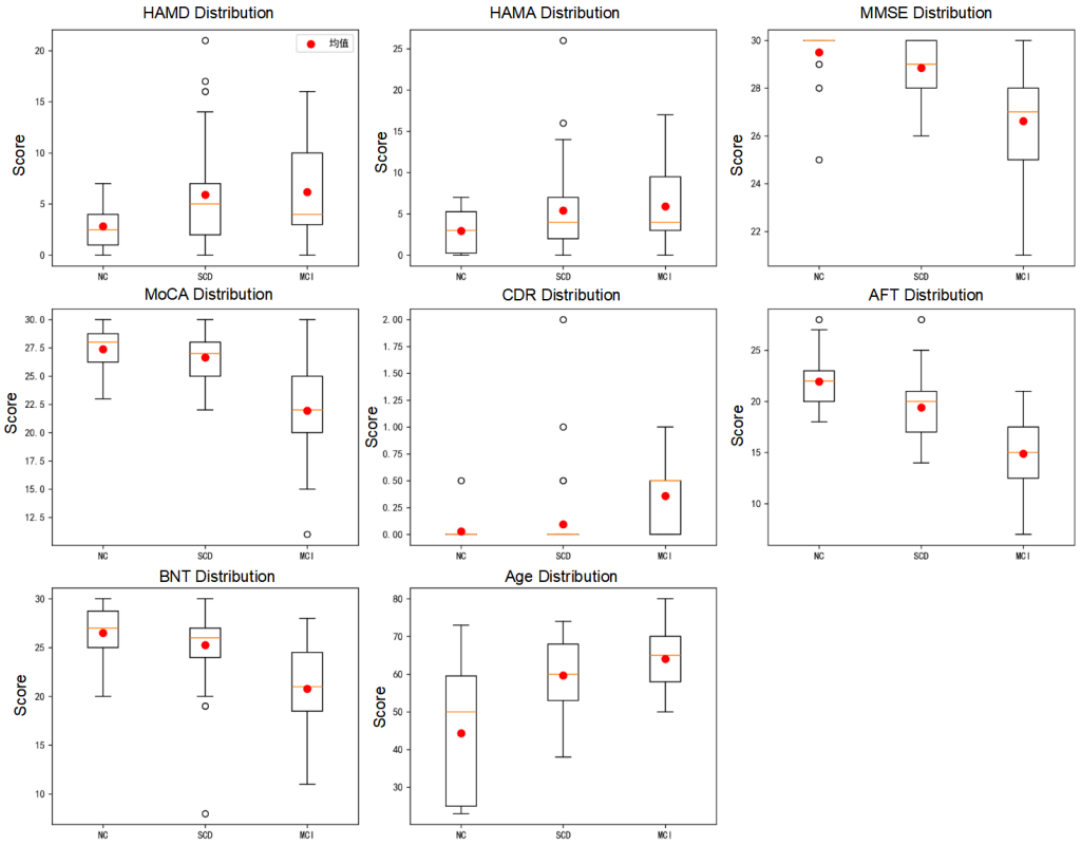


**Figure 1.** Distribution results at different clinical scales

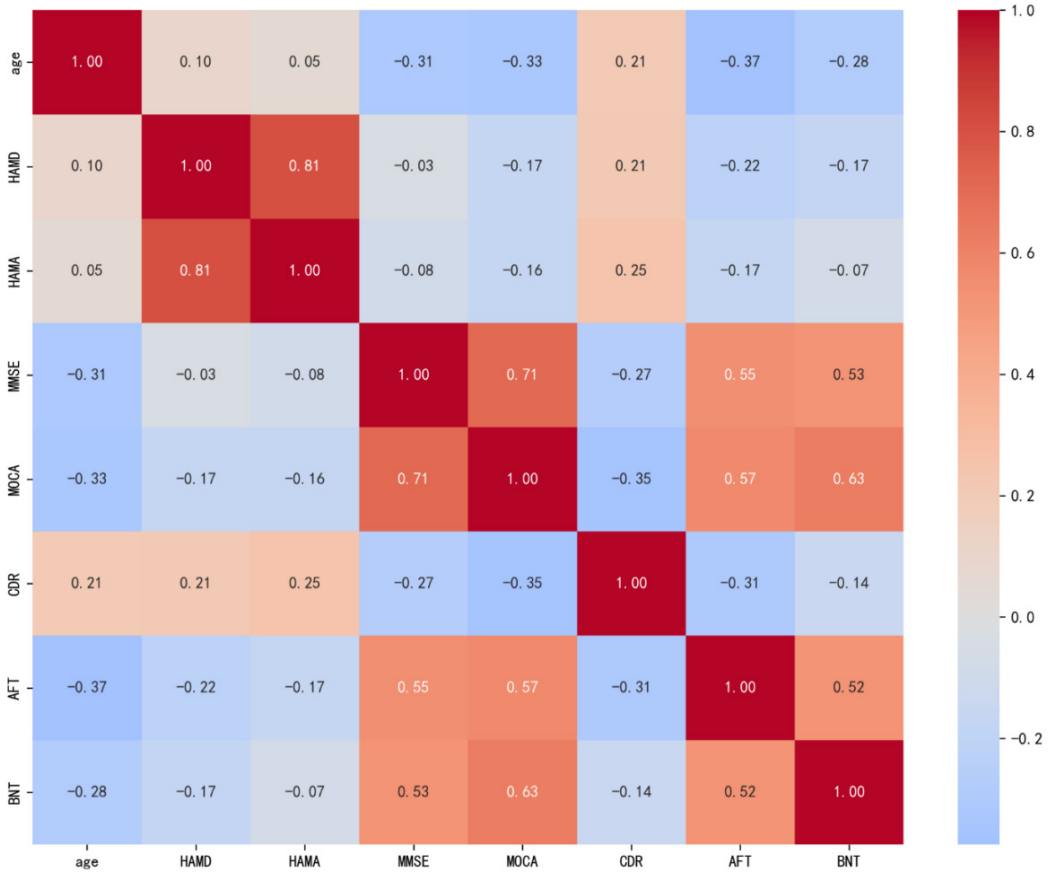


**Figure 2.** Correlation results at different clinical features

**Table 3.** Key correlation results between scales and age

| Variable 1 | Variable 2 | Correlation Coefficient (r) | p-value | Association Direction |
|---|---|---|---|---|
| HAMD | HAMA | 0.81 | <0.001 | Strong positive |
| MMSE | MoCA | 0.71 | <0.001 | Strong positive |
| MMSE | AFT | 0.55 | <0.001 | Moderate positive |
| MoCA | BNT | 0.63 | <0.001 | Moderate positive |
| Age | MMSE | -0.31 | 0.002 | Weak negative |
| Age | MoCA | -0.33 | 0.001 | Weak negative |
| CDR | MoCA | -0.35 | <0.001 | Weak negative |

## 4. Discussion

The findings of this study demonstrate notable disparities in all scale scores among the NC, SCD, and MCI populations, bearing distinct clinical and pathological implications. In the SCD group, self-reported cognitive decline was accompanied by early objective cognitive alterations (lower MoCA and AFT scores), which contradicts the notion of SCD as a "purely subjective" condition. This is consistent with the findings of Jessen et al. (2014), and pathological research provides corroboration: Positron emission tomography (PET) imaging reveals that 30-50% of individuals with SCD exhibit cerebral β - amyloid deposition, which shows a significant correlation with reduced MoCA scores [5]. This implies that SCD is not a benign state but rather a pre-clinical phase of cognitive impairment, necessitating early screening and monitoring. In the MCI group, significantly higher scores on the emotional scale compared to the SCD and NC groups indicate an interaction between cognitive impairment and emotional dysfunction. Cognitive decline impairs daily coping capabilities, leading to depression and anxiety, while emotional disorders accelerate cognitive deterioration (the hippocampal atrophy rate in depressed individuals is twice as high as that in cognitively intact individuals, and the hippocampus plays a central role in memory function [6]). Clinically, this underscores the necessity of simultaneous cognitive intervention and management of emotional symptoms in the MCI population. The CDR score of 0.5 only in the MCI group validates its value in differentiating MCI from SCD. However, due to its subjectivity (relying on the rater's judgment of daily function), it is essential to combine it with objective scales (e.g., MOCA, AFT) to enhance diagnostic accuracy.

A single screening scale exhibits significant limitations in the early identification of cognitive impairment. The MMSE demonstrates insufficient sensitivity for SCD, while the MoCA shows markedly reduced specificity in populations with lower education levels [7]. To address this, this study proposes a combined screening protocol for SCD: For community residents aged 50+ reporting cognitive decline, the initial MoCA assessment (those scoring <26 points proceed to the next stage) is followed by a verbal fluency test (AFT) (those scoring <20 points are deemed positive for screening). This two-stage progressive screening mechanism effectively identifies early cognitive changes and provides critical evidence for further evaluation of MCI. By integrating the comprehensive assessment advantages of MoCA across multiple cognitive domains with the high sensitivity of AFT for executive functions, this protocol significantly enhances the accuracy and efficiency of SCD identification, offering a feasible approach for early prevention and intervention of cognitive impairment in the community.

Comparisons with existing research show minor discrepancies. The HAMD scores in the SCD group (5.1±2.4) were higher than those reported by Smith et al. (2023) (3.8±2.1), presumably due to the higher proportion of community-dwelling participants in this study (who have less access to medical resources and greater psychological stress resulting from self-perceived cognitive decline). The MoCA scores in the MCI group (23.5±2.1) are consistent with the results of multi-center domestic studies [8], confirming the applicability of the MoCA across diverse Chinese populations. However, it is worth noting that the MMSE scores in our MCI group (26.2±1.8) were slightly lower than those reported in some international studies. This difference may be attributed to variations in educational background among the participants, as education level has been shown to influence MMSE performance. Additionally, the inclusion criteria for MCI in our study might have been more stringent, leading to the recruitment of a more cognitively impaired subgroup. Despite these minor discrepancies, our findings generally align with the existing literature, reinforcing the validity of our research methodology and the reliability of our results.

This study has several limitations. It is a single-center study with potential selection bias (participants from a tertiary hospital), lacks biological markers (e.g., brain imaging, cerebrospinal fluid) to verify the pathology of SCD, and employs a cross - sectional design that cannot capture dynamic changes in scale scores. Future research should incorporate multi - center samples, integrate biological markers to develop prediction models for the progression from SCD to MCI, and develop SCD-specific scales tailored to the Chinese population to improve the sensitivity and specificity of screening. Moreover, the relatively small sample size in this study may limit the generalizability of the findings. The exclusion criteria might have also led to the omission of some relevant cases, which could have provided more comprehensive insights. In terms of the assessment tools used, although they are widely recognized, there is still room for improvement in terms of their cultural adaptability for the Chinese context. Additionally, the follow-up period in this study was not long enough to fully observe the long-term progression patterns of SCD. To address these issues, subsequent studies could expand the sample size, relax some of the exclusion criteria under reasonable conditions, optimize the assessment tools, and extend the follow - up time to obtain more in - depth and reliable research results.

## 5. Conclusions

This research, by utilizing a comprehensive set of integrated multi-domain neuropsychological assessment scales, systematically investigates and elucidates the distinct clinical profiles and characteristics observed across populations with NC, SCD, and MCI. Notably, the findings indicate that individuals in the SCD group exhibit measurable and early-stage objective cognitive deficits-specifically reflected in (decreased scores on both the MoCA and the AFT-rather than experiencing solely subjective complaints of cognitive deterioration. In contrast, the MCI group displays a more complex clinical picture characterized by a significant interaction between progressive cognitive impairment and comorbid emotional dysregulation, which underscores the importance of implementing simultaneous and integrated interventions targeting both domains. Furthermore, the study reveals that the combined application of MoCA and AFT significantly enhances the accuracy and sensitivity of early SCD identification, providing a more reliable tool for differentiating it from normal aging or other pre-clinical states. Together, these insights contribute a solid quantitative and empirical foundation for improving early screening protocols and personalized intervention strategies for cognitive disorders, thereby actively supporting and advancing primary prevention efforts aimed at mitigating the onset and progression of AD.

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